KAL 03-06-00

=> s e6 or e5 or e1 or e2

L1 56 "WOLPERT ELISABETH Z"/AU OR "WOLPERT ELISABETH"/AU OR "WOLPERT E B"/AU OR "WOLPERT E Z"/AU

=> dup rem l1

PROCESSING COMPLETED FOR L1
L2 30 DUP REM L1 (26 DUPLICATES REMOVED)

=> d l2 ibib tot

L2 ANSWER 1 OF 30 MEDLINE
ACCESSION NUMBER: 1999367479 MEDLINE
DOCUMENT NUMBER: 99367479
TITLE: Vascular endothelial growth factor induces rapid phosphorylation of tight junction proteins occludin and zonula occluden 1. A potential mechanism for vascular permeability in diabetic retinopathy and tumors.
AUTHOR: Antonetti D A; Barber A J; Hollinger L A; **Wolpert E B**; Gardner T W
CORPORATE SOURCE: Penn State Retina Research Group, Pennsylvania State University College of Medicine, Hershey, Pennsylvania 17033, USA.. dantonetti@psu.edu
CONTRACT NUMBER: RO1 EY/DK12021 (NEI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 Aug 13) 274 (33) 23463-7.
Journal code: HIV. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals
ENTRY MONTH: 199911
ENTRY WEEK: 19991101

L2 ANSWER 2 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 1
ACCESSION NUMBER: 1999:355462 BIOSIS
DOCUMENT NUMBER: PREV199900355462
TITLE: T cell competition for the antigen-presenting cell as a model for immunodominance in the cytotoxic T lymphocyte response against minor histocompatibility antigens.
AUTHOR(S): Grufman, Per (1); **Wolpert, Elisabeth Z.**; Sandberg, Johan K.; Karre, Klas
CORPORATE SOURCE: (1) Microbiology and Tumor Biology Center, Karolinska Institutet, S-171 77, Stockholm Sweden
SOURCE: European Journal of Immunology, (July, 1999) Vol. 29, No. 7, pp. 2197-2204.
ISSN: 0014-2980.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

L2 ANSWER 3 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1999:274885 BIOSIS
DOCUMENT NUMBER: PREV199900274885
TITLE: Growth factor regulation of tight junction permeability in MDCK cells.
AUTHOR(S): Seese, N. K. (1); **Wolpert, E. B. (1)**; Barber, A. J. (1); Antonetti, D. A. (1)

CORPORATE SOURCE: (1) Penn State University College of Medicine, Hershey, PA U[redacted]
SOURCE: F[redacted]EB Journal, (March 15, 1999) Vo[redacted] 13, No. 5 PART 2, pp. A682.
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology 99 Washington, D.C., USA
April 17-21, 1999 Federation of American Societies for Experimental Biology
. ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English

L2 ANSWER 4 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1999:263823 BIOSIS
DOCUMENT NUMBER: PREV199900263823
TITLE: Mechanisms of apoptosis in the ischemic retina.
AUTHOR(S): Gardner, T. W. (1); **Wolpert, E. B. (1)**; Holinger, L. A.; Antonetti, D. A.; Barber, A. J. (1)
CORPORATE SOURCE: (1) Department of Ophthalmology, Penn State University College of Medicine, Hershey, PA USA
SOURCE: IOVS, (March 15, 1999) Vol. 40, No. 4, pp. S480.
Meeting Info.: Annual Meeting of the Association for Research in Vision and Ophthalmology Fort Lauderdale, Florida, USA May 9-14, 1999 Association for Research in Vision and Opthalmology
.
DOCUMENT TYPE: Conference
LANGUAGE: English

L2 ANSWER 5 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 2
ACCESSION NUMBER: 1999:417103 BIOSIS
DOCUMENT NUMBER: PREV199900417103
TITLE: Immunization with dendritic cells breaks immunodominance in
CTL responses against minor histocompatibility and synthetic peptide antigens.
AUTHOR(S): Grufman, Per (1); Sandberg, Johan K.; **Wolpert, Elisabeth Z.**; Karre, Klas
CORPORATE SOURCE: (1) Microbiology and Tumor Biology Center, Karolinska Institutet, Doktorsringen 13, S-171 77, Stockholm Sweden
SOURCE: Journal of Leukocyte Biology, (Aug., 1999) Vol. 66, No. 2, pp. 268-271.
ISSN: 0741-5400.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

L2 ANSWER 6 OF 30 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:402336 CAPLUS
DOCUMENT NUMBER: 129:80624
TITLE: Therapeutic applications of antigens or epitopes associated with impaired cellular peptide processing, e.g. expressed on RMA-S cells transfected with a B7-1 gene
INVENTOR(S): **Wolpert, Elisabeth**; Karre, Klas; Pettersson, Max; Sandberg, Johan
PATENT ASSIGNEE(S): Karolinska Innovation AB, Swed.; Wolpert, Elisabeth; Karre, Klas; Pettersson, Max; Sandberg, Johan
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9825645	A1	19980618	WO 1997-SE2094	19971212
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9854238	A1	19980703	AU 1998-54238	19971212
EP 964697	A1	19991222	EP 1997-948102	19971212
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
PRIORITY APPLN. INFO.:			SE 1996-4581	19961212
			WO 1997-SE2094	19971212

L2 ANSWER 7 OF 30 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1999008505 MEDLINE
DOCUMENT NUMBER: 99008505
TITLE: Immunodominance in the CTL response against minor histocompatibility antigens: interference between responding T cells, rather than with presentation of epitopes.
AUTHOR: **Wolpert E Z**; Grufman P; Sandberg J K; Tegnesjo A; Karre K
CORPORATE SOURCE: Microbiology and Tumor Biology Center, Karolinska Institute, Stockholm, Sweden.. elisabeth.wolpert@mtc.ki.se
SOURCE: JOURNAL OF IMMUNOLOGY, (1998 Nov 1) 161 (9) 4499-505. Journal code: IFB. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer Journals
ENTRY MONTH: 199901
ENTRY WEEK: 19990104

L2 ANSWER 8 OF 30 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 1998189758 MEDLINE
DOCUMENT NUMBER: 98189758
TITLE: Superdominance among immunodominant H-2Kb-restricted epitopes and reversal by dendritic cell-mediated antigen delivery.
AUTHOR: Sandberg J K; Grufman P; **Wolpert E Z**; Franksson L; Chambers B J; Karre K
CORPORATE SOURCE: Microbiology and Tumor Biology Center, Karolinska Institute, Stockholm, Sweden.. johan.sandberg@mtc.ki.se
SOURCE: JOURNAL OF IMMUNOLOGY, (1998 Apr 1) 160 (7) 3163-9. Journal code: IFB. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer Journals
ENTRY MONTH: 199806

L2 ANSWER 9 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 5
ACCESSION NUMBER: 1998:511549 BIOSIS
DOCUMENT NUMBER: PREV199800511549
TITLE: HA-1 and the SMCY-derived peptide FIDSYICQV (H-Y) are immunodominant minor histocompatibility antigens after bone marrow transplantation.
AUTHOR(S): Rufer, Nathalie; **Wolpert, Elisabeth**; Helg,

Claudine; Tiercy, Jean-Marie; Gratwohl, Alois; Chapuis, [redacted]nard; Jeannet, Michel; Goulmy, [redacted]s; Roosnek, Eddy (1)

CORPORATE SOURCE: [redacted] Unite d'Immunol. Transplantati[redacted], Hopital Cantonal Univ. Geneve, 24 rue Micheli-du-Crest, CH-1211 Geneve 14 Switzerland

SOURCE: Transplantation (Baltimore), (Oct. 15, 1998) Vol. 66, No. 7, pp. 910-916.
ISSN: 0041-1337.

DOCUMENT TYPE: Article
LANGUAGE: English

L2 ANSWER 10 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1998:203100 BIOSIS
DOCUMENT NUMBER: PREV199800203100
TITLE: Lung tumor formation is stimulated during compensatory lung
growth in 3-methyl-cholanthrene (MCA) treated mice.
AUTHOR(S): Brown, L. M. (1); **Wolpert, E. B.**; Rannels, D. E.; Malkinson, A. M.; Rannels, S. R.
CORPORATE SOURCE: (1) Pa. State Univ., Coll. Med., Hershey, PA 17033 USA
SOURCE: FASEB Journal, (March 20, 1998) Vol. 12, No. 5, pp. A812.
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology 98, Part II San Francisco, California, USA April 18-22, 1998 Federation of American Societies for Experimental Biology
. ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English

L2 ANSWER 11 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 6
ACCESSION NUMBER: 1997:501932 BIOSIS
DOCUMENT NUMBER: PREV199799801135
TITLE: Generation of CD8+ T cells specific for transporter associated with antigen processing deficient cells.
AUTHOR(S): **Wolpert, Elisabeth Z. (1)**; Peterson, Max; Chambers, Benedict J.; Sandberg, Johan K.; Kiessling, Rolf;
Ljunggren, Hans-Gustaf; Karre, Klas
CORPORATE SOURCE: (1) Microbiol. Tumor Biol. Cent., Karolinska Inst., Box 280, S-171 77 Stockholm Sweden
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 21, pp. 11496-11501.
ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English

L2 ANSWER 12 OF 30 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 96216090 MEDLINE
DOCUMENT NUMBER: 96216090
TITLE: The testis isoform of the phosphorylase kinase catalytic subunit (PhK-gammaT) plays a critical role in regulation of
glycogen mobilization in developing lung.
AUTHOR: Liu L; Rannels S R; Falconieri M; Phillips K S; **Wolpert E B**; Weaver T E
CORPORATE SOURCE: Division of Pulmonary Biology, Children's Hospital Medical Center, Cincinnati, Ohio 45229-3039, USA.
CONTRACT NUMBER: HD20748 (NICHD)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1996 May 17) 271 (20) 11761-6.
Journal code: HIV. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Cancer Journals

ENTRY MONTH: 199610

L2 ANSWER 13 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 8
ACCESSION NUMBER: 1995:392796 BIOSIS
DOCUMENT NUMBER: PREV199598407096
TITLE: Dominant and cryptic antigens in the MHC class I restricted
T cell response across a complex minor histocompatibility barrier: Analysis and mapping by elution of cellular peptides.
AUTHOR(S): **Wolpert, Elisabeth (1)**; Franksson, Lars; Karre, Klas
CORPORATE SOURCE: (1) Microbiol. Tumor Biol. Cent., Karolinska Inst., Box 280, 171 77 Stockholm Sweden
SOURCE: International Immunology, (1995) Vol. 7, No. 6, pp. 919-928.
ISSN: 0953-8178.
DOCUMENT TYPE: Article
LANGUAGE: English

L2 ANSWER 14 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1995:326180 BIOSIS
DOCUMENT NUMBER: PREV199598340480
TITLE: Cell Surface expression of cryptic minor histocompatibility
antigens.
AUTHOR(S): **Wolpert, Elisabeth**; Asserhed, Johan Sandberg; Karre, Klas
CORPORATE SOURCE: Microbiol. Tumorbiol. Cent., Karolinska Inst., Stockholm Sweden
SOURCE: Journal of Cellular Biochemistry Supplement, (1995) Vol. 0,
No. 21A, pp. 110.
Meeting Info.: Keystone Symposium on Control and Manipulation of the Immune Response Taos, New Mexico, USA March 16-22, 1995
ISSN: 0733-1959.
DOCUMENT TYPE: Conference
LANGUAGE: English

L2 ANSWER 15 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1994:326203 BIOSIS
DOCUMENT NUMBER: PREV199497339203
TITLE: General and unique peptide-specificities exhibited by transporters associated with antigen processing (TAP) from mouse, rat, and human.
AUTHOR(S): Franksson, Lars; **Wolpert, Elisabeth**; Karre, Klas
CORPORATE SOURCE: Microbiol. Tumorbiol. Center, Lab. Tumor Biol., Karolinska Inst., S-17177 Stockholm Sweden
SOURCE: Journal of Cellular Biochemistry Supplement, (1994) Vol. 0,
No. 18D, pp. 332.
Meeting Info.: Keystone Symposium on Lymphocyte Activation Keystone, Colorado, USA April 10-17, 1994
ISSN: 0733-1959.
DOCUMENT TYPE: Conference
LANGUAGE: English

L2 ANSWER 16 OF 30 MEDLINE
ACCESSION NUMBER: 94027361 MEDLINE
DOCUMENT NUMBER: 94027361
TITLE: Matrix Gla protein mRNA expression in cultured type II pneumocytes.
AUTHOR: Rannels S R; Cancela M L; **Wolpert E B**; Price P A
CORPORATE SOURCE: Department of Cellular and Molecular Physiology, College of

Medicine, Pennsylvania State University, Hershey 17033..
CONTRACT NUMBER: [redacted]42482 (NHLBI)
[redacted]-25921 (NIAMS)
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (1993 Sep) 265 (3 Pt 1) L270-8.
Journal code: 3U8. ISSN: 0002-9513.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199401

L2 ANSWER 17 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1992:294377 BIOSIS
DOCUMENT NUMBER: BR43:6727
TITLE: MATRIX GLA PROTEIN MRNA EXPRESSION IN CULTURED TYPE II PNEUMOCYTES.
AUTHOR(S): RANNELS S R; CANCELA M L; **WOLPERT E B**; PRICE P A
CORPORATE SOURCE: PENNSYLVANIA STATE UNIV., HERSHEY, PA.
SOURCE: 1992 INTERNATIONAL CONFERENCE OF THE AMERICAN LUNG ASSOCIATION AND THE AMERICAN THORACIC SOCIETY, MIAMI BEACH,
FLORIDA, USA, MAY 17-20, 1992. AM REV RESPIR DIS, (1992) 145 (4 PART 2), A138.
CODEN: ARDSBL. ISSN: 0003-0805.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L2 ANSWER 18 OF 30 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1990:570083 CAPLUS
DOCUMENT NUMBER: 113:170083
TITLE: Transfection of .beta.2-microglobulin restores IFN-mediated protection from natural killer cell lysis
in YAC-1 lymphoma variants
AUTHOR(S): Ljunggren, Hans Gustaf; Sturmhoefel, Knut; **Wolpert, Elisabeth**; Haemmerling, Guenter J.; Kaerre, Klas
CORPORATE SOURCE: Dep. Tumor Biol., Karolinska Inst., Stockholm, S-104 01, Swed.
SOURCE: J. Immunol. (1990), 145(1), 380-6
CODEN: JOIMA3; ISSN: 0022-1767
DOCUMENT TYPE: Journal
LANGUAGE: English

L2 ANSWER 19 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1990:319575 BIOSIS
DOCUMENT NUMBER: BR39:26911
TITLE: EFFECT OF DIETARY SODIUM DEFICIENCY ON NORMAL AND COMPENSATORY GROWTH OF THE LUNG.
AUTHOR(S): GALLAHER K J; **WOLPERT E B**; WASSNER S J; RANNELS D E
CORPORATE SOURCE: DEP. PEDIATRICS, PENN STATE UNIV. COLL. MED., HERSHEY, PA. 17033.
SOURCE: WORLD CONFERENCE ON LUNG HEALTH, BOSTON, MASSACHUSETTS, USA, MAY 20-24, 1990. AM REV RESPIR DIS, (1990) 141 (4 PART 2), A344.
CODEN: ARDSBL. ISSN: 0003-0805.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L2 ANSWER 20 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 9
ACCESSION NUMBER: 1991:156936 BIOSIS

DOCUMENT NUMBER: BA91:82736
TITLE: [redacted]MA CARBOXYGLUTAMIC ACID EXCRETI[redacted] INTO RAT AMNIOTIC [redacted]ID DURING LATE GESTATION.
AUTHOR(S): GALLAHER K J; **WOLPERT E B**; RANNELS S R
CORPORATE SOURCE: DIV. NEONATOL., CAPE FEAR VALLEY MED. CENT., P.O. BOX 2000,
FAYETTEVILLE, N.C. 28302, USA.
SOURCE: J DEV PHYSIOL (EYNSHAM), (1990) 13 (6), 327-332.
CODEN: JDPHDH. ISSN: 0141-9846.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L2 ANSWER 21 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1990:256867 BIOSIS
DOCUMENT NUMBER: BR38:123455
TITLE: GAMMA CARBOXYGLUTAMIC ACID GLA EXCRETION IN RATS MADE SODIUM DEFICIENT BY DIET.
AUTHOR(S): GALLAHER K J; **WOLPERT E B**; RANNELS D E; WASSNER S J
CORPORATE SOURCE: PENN STATE UNIV. COLL. MED., MS HERSHEY MED. CENT., DEP. PEDIATR., HERSHEY, PA.
SOURCE: JOINT MEETING OF THE AMERICAN PEDIATRIC SOCIETY AND THE SOCIETY FOR PEDIATRIC RESEARCH, ANAHEIM, CALIFORNIA, USA, MAY 7-10, 1990. PEDIATR RES, (1990) 27 (4 PART 2), 283A.
CODEN: PEREBL. ISSN: 0031-3998.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L2 ANSWER 22 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1991:43462 BIOSIS
DOCUMENT NUMBER: BR40:20442
TITLE: VITAMIN K-DEPENDENT CARBOXYLATION IN RAT LUNG AND ISOLATED TYPE II PNEUMOCYTES.
AUTHOR(S): RANNELS S R; FERNSLER R D; **WOLPERT E B**; GALLAHER K J
CORPORATE SOURCE: DEP. CELLULAR MOLECULAR PHYSIOL., PENNSYLVANIA STATE UNIV. COLL. MED., HERSHEY, PA. 17033.
SOURCE: THIRTIETH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR CELL BIOLOGY, SAN DIEGO, CALIFORNIA, USA, DECEMBER 9-13, 1990.
J
CELL BIOL, (1990) 111 (5 PART 2), 55A.
CODEN: JCLBA3. ISSN: 0021-9525.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L2 ANSWER 23 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 10
ACCESSION NUMBER: 1988:423510 BIOSIS
DOCUMENT NUMBER: BA86:86122
TITLE: MINIMIZING PERIOPERATIVE HYPOXEMIA DOES NOT AFFECT POSTPNEUMONECTOMY LUNG GROWTH.
AUTHOR(S): KARL H W; **WOLPERT E B**; RANNELS D E
CORPORATE SOURCE: DEP. ANESTHESIA, MILTON S. HERSHEY MED. CENT., PENNSYLVANIA
STATE UNIV., HERSHEY, PA. 17033.
SOURCE: AM J PHYSIOL, (1988) 255 (1 PART 1), E65-E69.
CODEN: AJPHAP. ISSN: 0002-9513.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L2 ANSWER 24 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 11
ACCESSION NUMBER: 1971:172632 BIOSIS
DOCUMENT NUMBER: BA52:82632
TITLE: REGULATION OF PROTEIN SYNTHESIS IN HEART MUSCLE PART 3 EFFECT OF ANOXIA ON PROTEIN SYNTHESIS.

AUTHOR(S): JEFFERSON L S; **WOLPERT E B**; GIGER K E; MORGAN H E
SOURCE: J BIOL CHEM, (1971) 246 (7), 2171-2178.
CODEN: JBCHA3. ISSN: 0021-9258.
FILE SEGMENT: BA; OLD
LANGUAGE: Unavailable

L2 ANSWER 25 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 12
ACCESSION NUMBER: 1971:172631 BIOSIS
DOCUMENT NUMBER: BA52:82631
TITLE: REGULATION OF PROTEIN SYNTHESIS IN HEART MUSCLE PART 2 EFFECT OF AMINO-ACID LEVELS AND INSULIN ON RIBOSOMAL AGGREGATION.
AUTHOR(S): MORGAN H E; JEFFERSON L S; **WOLPERT E B**; RANNELS D E
SOURCE: J BIOL CHEM, (1971) 246 (7), 2163-2170.
CODEN: JBCHA3. ISSN: 0021-9258.
FILE SEGMENT: BA; OLD
LANGUAGE: Unavailable

L2 ANSWER 26 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 13
ACCESSION NUMBER: 1971:172630 BIOSIS
DOCUMENT NUMBER: BA52:82630
TITLE: REGULATION OF PROTEIN SYNTHESIS IN HEART MUSCLE PART 1 EFFECT OF AMINO-ACID LEVELS ON PROTEIN SYNTHESIS.
AUTHOR(S): MORGAN H E; EARL D C N; BROADUS A; **WOLPERT E B**; GIGER K E; JEFFERSON L S
SOURCE: J BIOL CHEM, (1971) 246 (7), 2152-2162.
CODEN: JBCHA3. ISSN: 0021-9258.
FILE SEGMENT: BA; OLD
LANGUAGE: Unavailable

L2 ANSWER 27 OF 30 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1971:507216 CAPLUS
DOCUMENT NUMBER: 75:107216
TITLE: Effects of anoxia on protein synthesis in heart muscle
AUTHOR(S): Jefferson, L. S.; **Wolpert, E. B.**; Giger, K. E.; Forney, L. W.; Morgan, Howard E.
CORPORATE SOURCE: Coll. Med., Pennsylvania State Univ., Philadelphia, Pa., USA
SOURCE: Cardiac Hypertrophy (1971), 345-54. Editor(s): Alpert, Norman R. Academic: New York, N. Y.
CODEN: 23VRA5
DOCUMENT TYPE: Conference
LANGUAGE: English

L2 ANSWER 28 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 14
ACCESSION NUMBER: 1971:27669 BIOSIS
DOCUMENT NUMBER: BR07:27669
TITLE: MAINTENANCE OF PROTEIN SYNTHESIS IN HEARTS OF DIABETIC ANIMALS.
AUTHOR(S): RANNELS D E; JEFFERSON L S; HJALMARSON A C; **WOLPERT E B**; MORGAN H E
SOURCE: Biochem. Biophys. Res. Commun., (1970) 40 (5), 1110-1116.
CODEN: BBRCA9. ISSN: 0006-291X.
FILE SEGMENT: BR; OLD
LANGUAGE: Unavailable

L2 ANSWER 29 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1970:39926 BIOSIS
DOCUMENT NUMBER: BR06:39926
TITLE: PROTEIN SYNTHESIS IN HEART AFTER HYPOPHYSECTOMY.
AUTHOR(S): HJALMARSON A C; GIGER K E; **WOLPERT E B**; MORGAN H E
SOURCE: Fed. Proc., (1970) 29 (2), 580.
CODEN: FEPRA7. ISSN: 0014-9446.

DOCUMENT TYPE: Conference
FILE SEGMENT: [redacted] OLD
LANGUAGE: [redacted]vailable

L2 ANSWER 30 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1969:46732 BIOSIS
DOCUMENT NUMBER: BR05:46732
TITLE: REGULATION OF PROTEIN SYNTHESIS IN HEART MUSCLE ABSTRACT.
AUTHOR(S): MORGAN H E; **WOLPERT E B**; GIGER K E
SOURCE: Fed. Proc., (1969) 28 (2), 782.
CODEN: FEPRA7. ISSN: 0014-9446.
FILE SEGMENT: BR; OLD
LANGUAGE: Unavailable

=> d l2 18 15 14 13 11 9 7 5 2 ibib abs

L2 ANSWER 18 OF 30 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1990:570083 CAPLUS
DOCUMENT NUMBER: 113:170083
TITLE: Transfection of .beta.2-microglobulin restores IFN-mediated protection from natural killer cell lysis in YAC-1 lymphoma variants
AUTHOR(S): Ljunggren, Hans Gustaf; Sturmhoefel, Knut; **Wolpert, Elisabeth**; Haemmerling, Guenter J.; Kaerre, Klas
CORPORATE SOURCE: Dep. Tumor Biol., Karolinska Inst., Stockholm, S-104 01, Swed.
SOURCE: J. Immunol. (1990), 145(1), 380-6
CODEN: JOIMA3; ISSN: 0022-1767
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A .beta.2-microglobulin (.beta.2m)-deficient variant of YAC-1, A.H-2-, was transfected with a genomic .beta.2m clone. Transfected cells were used to investigate the role of .beta.2m in IFN-induced protection from natural killer (NK) cell lysis. IFN-.gamma. lymphoma results in reduced sensitivity to NK cell-mediated lysis in parallel with increased expression of its constitutively low MHC class I expression. It was previously shown that the A.H-2- variant had lost both these capacities, although it retained other responses to IFN-.gamma.. Here .beta.2m transfection restored the YAC-1 phenotype with respect to an inducible expression of MHC class I mols. and a concomitant protection from NK cell lysis after treatment with IFN-.gamma.. In the absence of IFN-.gamma. the NK sensitivity of the transfectants did not differ significantly from A.H-2-. A similar protection from NK cell lysis, in parallel with enhanced MHC class I expression, was obsd. for in vivo-passaged .beta.2m transfectants whereas no protection was found for in vivo-passaged A.H-2- cells. Thus, the IFN-.gamma.-mediated protection from NK cell lysis is dependent on .beta.2m expression in the YAC-1 lymphoma. Restoration of MHC class I assembly, transport, and concomitantly an IFN-.gamma. augmentable cell surface expression of MHC class I mols. is a possible explanation for the effect of .beta.2m.

L2 ANSWER 15 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1994:326203 BIOSIS
DOCUMENT NUMBER: PREV199497339203
TITLE: General and unique peptide-specificities exhibited by transporters associated with antigen processing (TAP) from mouse, rat, and human.
AUTHOR(S): Franksson, Lars; **Wolpert, Elisabeth**; Karre, Klas
CORPORATE SOURCE: Microbiol. Tumorbiol. Center, Lab. Tumor Biol., Karolinska

Inst., S-17177 Stockholm Sweden
SOURCE: Journal of Cellular Biochemistry Supplement, (1994) Vol. 0,
No. 18D, pp. 332.
Meeting Info.: Keystone Symposium on Lymphocyte Activation Keystone, Colorado, USA April 10-17, 1994
ISSN: 0733-1959.
DOCUMENT TYPE: Conference
LANGUAGE: English

L2 ANSWER 14 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS
ACCESSION NUMBER: 1995:326180 BIOSIS
DOCUMENT NUMBER: PREV199598340480
TITLE: Cell Surface expression of cryptic minor histocompatibility antigens.
AUTHOR(S): **Wolpert, Elisabeth**; Asserhed, Johan Sandberg; Karre, Klas
CORPORATE SOURCE: Microbiol. Tumorbiol. Cent., Karolinska Inst., Stockholm Sweden
SOURCE: Journal of Cellular Biochemistry Supplement, (1995) Vol. 0,
No. 21A, pp. 110.
Meeting Info.: Keystone Symposium on Control and Manipulation of the Immune Response Taos, New Mexico, USA March 16-22, 1995
ISSN: 0733-1959.
DOCUMENT TYPE: Conference
LANGUAGE: English

L2 ANSWER 13 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 8
ACCESSION NUMBER: 1995:392796 BIOSIS
DOCUMENT NUMBER: PREV199598407096
TITLE: Dominant and cryptic antigens in the MHC class I restricted T cell response across a complex minor histocompatibility barrier: Analysis and mapping by elution of cellular peptides.
AUTHOR(S): **Wolpert, Elisabeth (1)**; Franksson, Lars; Karre, Klas
CORPORATE SOURCE: (1) Microbiol. Tumor Biol. Cent., Karolinska Inst., Box 280, 171 77 Stockholm Sweden
SOURCE: International Immunology, (1995) Vol. 7, No. 6, pp. 919-928.
ISSN: 0953-8178.
DOCUMENT TYPE: Article
LANGUAGE: English
AB T cell responses against complex antigens are often directed against a limited set of immunodominant determinants. We have studied this phenomenon at the level of cellularly processed peptides recognized by CTL in the B6 anti-BALB.B minor histocompatibility (H) barrier, comprising at least 29 antigen loci. B6 anti-BALB.B CTL always recognized three reverse phase HPLC fractions in BALB.B eluates, whether the latter were obtained from cell lysates or immunoaffinity purified class I molecules. One of these immunodominant epitopes (termed IDE-1) was H-2D-b restricted, and two (termed IDE-2 and IDE-3) were H-2K-b restricted. B6 mice were immunized with spleen cells from B6 congenic mice carrying single minor H loci from BALB.B with the aim to assign IDE to given minor H loci and to investigate whether additional epitopes could be identified in the absence of the immunodominant ones. IDE-3 was found to be associated to the locus H-28; in addition five so called cryptic epitopes were defined. Induction of CTL against these epitopes required immunization with cells of the congenic strain; BALB.B spleen cells failed to immunize. One subgroup of these epitopes (those associated to H-8, H-19 and H-25) were nevertheless

found to be processed and loaded in class I molecules of BALB.B cells, while there was [redacted] evidence for this for H-35 and [redacted]36. For 10 additional congenic strains, no CTL response was detected. The results are discussed in relation to the genetic and molecular basis of minor H antigens, and mechanisms for epitope dominance operating at the level of the APC or responding T cells.

L2 ANSWER 11 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 6
ACCESSION NUMBER: 1997:501932 BIOSIS
DOCUMENT NUMBER: PREV199799801135
TITLE: Generation of CD8+ T cells specific for transporter associated with antigen processing deficient cells.
AUTHOR(S): **Wolpert, Elisabeth Z. (1)**; Peterson, Max; Chambers, Benedict J.; Sandberg, Johan K.; Kiessling, Rolf; Ljunggren, Hans-Gustaf; Karre, Klas
CORPORATE SOURCE: (1) Microbiol. Tumor Biol. Cent., Karolinska Inst., Box 280, S-171 77 Stockholm Sweden
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 21, pp. 11496-11501.
ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Cells with impaired transporter associated with antigen processing (TAP) function express low levels of cell surface major histocompatibility complex (MHC) class I molecules, and are generally resistant to lysis by MHC class I restricted cytotoxic T lymphocytes (CTLs). Here we report the generation of MHC class I restricted CD8+ CTLs that surprisingly require target cell TAP deficiency for efficient recognition. C57BL/6 (B6) mice immunized with syngenic B7-1 (CD80) expressing TAP-deficient cells generated a potent CTL response against both TAP-deficient RMA-S tumor cells and TAP-deficient Con A blasts, whereas the corresponding TAP-expressing target cells were considerably less susceptible or resistant to lysis. The CTL epitopes recognized were expressed also by the human TAP-deficient cell line T2, transfected with appropriate MHC class I molecules. B6 mice immunized with B7-1-transfected TAP-deficient RMA-S cells were protected from outgrowth of a subsequent RMA-S tumor challenge. These findings are discussed in relation to the biochemical nature of MHC class I dependent CTL epitopes associated with impaired TAP function, as well as implications for immunotherapy and autoimmunity.

L2 ANSWER 9 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 5
ACCESSION NUMBER: 1998:511549 BIOSIS
DOCUMENT NUMBER: PREV199800511549
TITLE: HA-1 and the SMCY-derived peptide FIDSYICQV (H-Y) are immunodominant minor histocompatibility antigens after bone marrow transplantation.
AUTHOR(S): Rufer, Nathalie; **Wolpert, Elisabeth**; Helg, Claudine; Tiercy, Jean-Marie; Gratwohl, Alois; Chapuis, Bernard; Jeannet, Michel; Goulmy, Els; Roosnek, Eddy (1)
CORPORATE SOURCE: (1) Unite d'Immunol. Transplantation, Hopital Cantonal Univ. Geneve, 24 rue Micheli-du-Crest, CH-1211 Geneve 14 Switzerland
SOURCE: Transplantation (Baltimore), (Oct. 15, 1998) Vol. 66, No. 7, pp. 910-916.
ISSN: 0041-1337.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Background. Allogeneic bone marrow donors can be incompatible at different levels. Even HLA-identical pairs will be still incompatible for numerous

minor histocompatibility antigens (mHag). Nevertheless, some incompatibilities are found to be associated with an increased risk of graft-versus-host disease (GVHD), which could be related to the way the immune system recognizes these antigens. Methods. We determined the specificity of cytotoxic T-cell clones isolated during acute GVHD or during bone marrow graft rejection in patients (n=14) transplanted with marrow from donors who were histoincompatible for different minor and/or major histocompatibility antigens. Results. We found a clear hierarchy among the different types of histoincompatibilities. In three combinations mismatched for a class I allele, all 27 clones isolated during GVHD were specific for the incompatible HIA molecule. In the 11 class I-identical combinations, 14 different mHags were recognized. The mHag HA-1, known to have a significant impact on the development of GVHD, was recognized in the two HA-1incompatible combinations. In one of these combinations, which was sex mismatched, all 56 clones analyzed were directed against HA-1, demonstrating the dominance of this mHag. In the four HA-1-compatible, sex-mismatched combinations, the anti-H-Y response was directed against one immunodominant epitope rather than against multiple Y-chromosome encoded epitopes. All male specific cytotoxic T lymphocytes (n=15) recognized the same high-performance liquid chromatography-purified peptide fraction presented by T2 cells. Moreover, all cytotoxic T lymphocytes tested (n = 6) were specific for the SMCY-derived peptide FIDSYICQV, originally described as being the H-Y epitope recognized in the context of HLA-A*0201. Conclusions. Some histocompatibility antigens are recognized in an immunodominant fashion and will therefore be recognized in the majority of mismatched combinations. Only for such antigens, correlations between mismatches and the occurrence of GVHD or graft rejections will be found.

L2 ANSWER 7 OF 30 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 1999008505 MEDLINE
DOCUMENT NUMBER: 99008505
TITLE: Immunodominance in the CTL response against minor histocompatibility antigens: interference between responding T cells, rather than with presentation of epitopes.
AUTHOR: **Wolpert E Z**; Grufman P; Sandberg J K; Tegnesjo A; Karre K
CORPORATE SOURCE: Microbiology and Tumor Biology Center, Karolinska Institute, Stockholm, Sweden.. elisabeth.wolpert@mtc.ki.se
SOURCE: JOURNAL OF IMMUNOLOGY, (1998 Nov 1) 161 (9) 4499-505. Journal code: IFB. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Cancer Journals
ENTRY MONTH: 199901
ENTRY WEEK: 19990104
AB We have investigated mechanisms involved in immunodominance of the CTL response of C57BL/6 (B6) mice against cells of BALB.B origin. This transplantation barrier consists of at least 40 minor histocompatibility (H) Ags. Insufficient presentation of nondominant epitopes in the presence of dominant epitopes was investigated as a possible mechanism for immunodominance. Ag presentation was assessed by recognition of dendritic cells of BALB.B origin, MLC restimulatory capacity, and quantification of cell surface presentation by peptide elution from intact cells. Cells from BALB.B mice, which fail to elicit CTL against nondominant epitopes, presented nondominant epitopes to a similar extent as cells from minor H congenic mice; the latter do elicit CTL against nondominant minor H Ags. Nevertheless, presentation of nondominant and dominant epitopes by the

same APC appeared to be an important factor for immunodominance to occur, since simultaneous immunization with the epitopes on separate cells elicited CTL against both types of epitopes. This suggested that immunodominance is determined in the interaction between different responding T cells and the APC. Support for this was obtained in an in vitro model in which the CTL response against a nondominant epitope was inhibited by the concomitant response against a dominant epitope. This study suggests that immunodominance in the CTL response against certain minor H Ags results from interference between T cell responses and not from insufficient presentation of peptide epitopes. The study also provides an in vitro model for further investigations of the immunodominance phenomenon.

L2 ANSWER 5 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 2
ACCESSION NUMBER: 1999:417103 BIOSIS
DOCUMENT NUMBER: PREV199900417103
TITLE: Immunization with dendritic cells breaks immunodominance in CTL responses against minor histocompatibility and synthetic peptide antigens.
AUTHOR(S): Grufman, Per (1); Sandberg, Johan K.; **Wolpert, Elisabeth Z.**; Karre, Klas
CORPORATE SOURCE: (1) Microbiology and Tumor Biology Center, Karolinska Institutet, Doktorsringen 13, S-171 77, Stockholm Sweden
SOURCE: Journal of Leukocyte Biology, (Aug., 1999) Vol. 66, No. 2, pp. 268-271.
ISSN: 0741-5400.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB We have examined the mechanisms involved in immunodominance in two different experimental models: the cytotoxic T lymphocyte (CTL) response in B6 mice against minor histocompatibility antigens of BALB.B mice, and the response of B6 mice against a mixture of five synthetic peptides corresponding to well-defined immunogenic epitopes. The CTL responses in these models focus on a few dominant epitopes, whereas no or only weak responses can be detected against other subdominant epitopes. Neither of these immunodominance phenomena can be explained by insufficient presentation of subdominant epitopes in the presence of the dominant ones.
Immunodominance could also be demonstrated in an in vitro system, in which B6 splenocytes primed with BALB.B could interfere with the CTL response against subdominant antigens. This interference was dependent on CD8+ T cells and on the simultaneous presentation of dominant and subdominant antigens on the same antigen-presenting cell, suggesting T cell competition around the antigen-presenting cell as a potential explanation.
The immunodominance in both systems could be broken by immunization with dendritic cells (from BALB.B or from B6 loaded with peptides). This procedure allowed detection of CTL responses against both dominant and previously subdominant antigens.

L2 ANSWER 2 OF 30 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 1
ACCESSION NUMBER: 1999:355462 BIOSIS
DOCUMENT NUMBER: PREV199900355462
TITLE: T cell competition for the antigen-presenting cell as a model for immunodominance in the cytotoxic T lymphocyte response against minor histocompatibility antigens.
AUTHOR(S): Grufman, Per (1); **Wolpert, Elisabeth Z.**; Sandberg, Johan K.; Karre, Klas
CORPORATE SOURCE: (1) Microbiology and Tumor Biology Center, Karolinska Institutet, S-171 77, Stockholm Sweden
SOURCE: European Journal of Immunology, (July, 1999) Vol. 29, No. 7, pp. 2197-2204.
ISSN: 0014-2980.

DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English

AB We recently demonstrated that spleen cells primed against dominant BALB.B antigens can inhibit the cytotoxic T lymphocyte (CTL) response against subdominant antigens in vitro. In this study, we show that this interference is dependent on CD8+, but not CD4+, T cells directed against dominant antigens. Similar to immunodominance in vivo, T cell interference in vitro required presentation of dominant and sobdominant antigens by the same antigen-presenting cell. In vivo priming with cells expressing dominant and subdominant antigens did not induce long-lasting unresponsiveness against the latter. These results support a model in which immunodominance is mediated by T cell competition. In line with this, we found that the immunodominance effects in the CTL response against these minor histocompatibility antigens could be broken by immunization with live bone marrow-derived dendritic cells.

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L3 1062 MHC AND CD80

=> s l3 and rma()s

L4 10 L3 AND RMA(W) S

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PROCESSING COMPLETED FOR L4
L5 4 DUP REM L4 (6 DUPLICATES REMOVED)

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L5 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:402336 CAPLUS
DOCUMENT NUMBER: 129:80624
TITLE: Therapeutic applications of antigens or epitopes associated with impaired cellular peptide processing, e.g. expressed on **RMA-S** cells transfected with a B7-1 gene
INVENTOR(S): Wolpert, Elisabeth; Karre, Klas; Pettersson, Max; Sandberg, Johan
PATENT ASSIGNEE(S): Karolinska Innovation AB, Swed.; Wolpert, Elisabeth; Karre, Klas; Pettersson, Max; Sandberg, Johan
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9825645	A1	19980618	WO 1997-SE2094	19971212
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9854238	A1	19980703	AU 1998-54238	19971212

EP 964697 A1 19991222 EP 1997-948102 19971212
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI
PRIORITY APPLN. INFO.: SE 1996-4581 19961212
WO 1997-SE2094 19971212

AB The present invention relates to use of substances that can induce expression of antigens or epitopes assocd. with impaired cellular peptide processing, esp. **MHC** class I dependent, for prepn. of pharmaceuticals, pharmaceutical compns. or vaccines, that stimulate specific T-cell mediated immune responses against cancer and virus infected cells. It also relates to use of antigens or epitopes assocd. with impaired cellular peptide processing, esp. **MHC** class I dependent, or part thereof, for the same purpose. It also relates to mammalian cells that have been manipulated to express antigens or epitopes assocd. with impaired cellular peptide processing, esp. **MHC** class I dependent, and to lymphoid cells activated against such **MHC** class I dependent structures for the same purpose. Furthermore, processes for such manipulation of mammalian cells and for treating human beings as well as kits for use in such manipulations are covered. The present invention also relates to use of mols. including T-cell receptors or part thereof directed against antigens or epitopes assocd. with impaired cellular peptide processing, esp. **MHC** class I dependent, for prepn. of pharmaceuticals, pharmaceutical compns. or vaccines. According to the present invention, the ultimate purpose of the products or processes above is the treatment, prevention and diagnosis of cancers and virus infections.

=> d l5 ibib abs tot

L5 ANSWER 1 OF 4 CAPLUS COPYRIGHT 2000 ACS
ACCESSION NUMBER: 1998:402336 CAPLUS
DOCUMENT NUMBER: 129:80624
TITLE: Therapeutic applications of antigens or epitopes associated with impaired cellular peptide processing, e.g. expressed on **RMA-S** cells transfected with a B7-1 gene
INVENTOR(S): Wolpert, Elisabeth; Karre, Klas; Pettersson, Max; Sandberg, Johan
PATENT ASSIGNEE(S): Karolinska Innovation AB, Swed.; Wolpert, Elisabeth; Karre, Klas; Pettersson, Max; Sandberg, Johan
SOURCE: PCT Int. Appl., 55 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9825645	A1	19980618	WO 1997-SE2094	19971212
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9854238	A1	19980703	AU 1998-54238	19971212
EP 964697	A1	19991222	EP 1997-948102	19971212
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: SE 1996-4581 19961212
WO 1997-SE20 19971212

AB The present invention relates to use of substances that can induce expression of antigens or epitopes assocd. with impaired cellular peptide processing, esp. **MHC** class I dependent, for prepn. of pharmaceuticals, pharmaceutical compns. or vaccines, that stimulate specific T-cell mediated immune responses against cancer and virus infected cells. It also relates to use of antigens or epitopes assocd. with impaired cellular peptide processing, esp. **MHC** class I dependent, or part thereof, for the same purpose. It also relates to mammalian cells that have been manipulated to express antigens or epitopes assocd. with impaired cellular peptide processing, esp. **MHC** class I dependent, and to lymphoid cells activated against such **MHC** class I dependent structures for the same purpose. Furthermore, processes for such manipulation of mammalian cells and for treating human beings as well as kits for use in such manipulations are covered. The present invention also relates to use of mols. including T-cell receptors or part thereof directed against antigens or epitopes assocd. with impaired cellular peptide processing, esp. **MHC** class I dependent, for prepn. of pharmaceuticals, pharmaceutical compns. or vaccines. According to the present invention, the ultimate purpose of the products or processes above is the treatment, prevention and diagnosis of cancers and virus infections.

L5 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 1
ACCESSION NUMBER: 1998:394179 BIOSIS
DOCUMENT NUMBER: PREV199800394179
TITLE: Combination therapy with fumagillin and vaccination with tumor-derived antigenic peptides in B16 melanoma-transplanted mice.
AUTHOR(S): Tsuji, Tumio (1); Yamada, Koichi
CORPORATE SOURCE: (1) Discovery Res. Div., Santen Pharm. Co. Ltd., 3-9-19 Shimoshinjo, Higashiyodogawa-ku, Osaka 533-8651 Japan
SOURCE: International Journal of Immunopharmacology, (Jan.-March, 1998) Vol. 20, No. 1-3, pp. 111-124.
ISSN: 0192-0561.
DOCUMENT TYPE: Article
LANGUAGE: English

AB We established the antigen-presenting cell line **RMA-S** /mCD80 expressing mouse **CD80**. **RMA-S** is an antigen processing-defective mutant originating from RMA lymphoma and can be loaded with exogenous immunogenic peptides on the major histocompatibility complex class I (**MHC**-I) molecules. After immunization with **RMA-S** or **RMA-S** /mCD80 loaded with B16 melanoma-derived peptides, only **RMA-S**/mCD80 pulsed with B16 melanoma-derived peptides showed antitumor effects against B16 melanoma in vivo. However, it showed little enhancement of survival. On the other hand, fumagillin, an inhibitor of angiogenesis, suppressed B16 melanoma growth and showed a survival promoting effect. Combination therapy with fumagillin and vaccination with B16 melanoma-derived peptides strongly inhibited tumor growth and promoted survival more than fumagillin therapy alone. These results suggest that vaccination with poorly immunogenic tumor-derived peptides combined with antitumor drugs, such as anti-angiogenic compounds, may be useful.

L5 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 2
ACCESSION NUMBER: 1997:501932 BIOSIS
DOCUMENT NUMBER: PREV199799801135
TITLE: Generation of CD8+ T cells specific for transporter associated with antigen processing deficient cells.
AUTHOR(S): Wolpert, Elisabeth Z. (1); Peterson, Max; Chambers, Benedict J.; Sandberg, Johan K.; Kiessling, Rolf;

CORPORATE SOURCE: Ljunggren, Hans-Gustaf; Karre, Klas; (I[redacted] Microbiol. Tumor Biol. Cent., K[redacted]olinska Inst., Box 280, S-171 77 Stockholm Sweden

SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1997) Vol. 94, No. 21, pp. 11496-11501.
ISSN: 0027-8424.

DOCUMENT TYPE: Article

LANGUAGE: English

AB Cells with impaired transporter associated with antigen processing (TAP) function express low levels of cell surface major histocompatibility complex (**MHC**) class I molecules, and are generally resistant to lysis by **MHC** class I restricted cytotoxic T lymphocytes (CTLs). Here we report the generation of **MHC** class I restricted CD8+ CTLs that surprisingly require target cell TAP deficiency for efficient recognition. C57BL/6 (B6) mice immunized with syngenic B7-1 (**CD80**) expressing TAP-deficient cells generated a potent CTL response against both TAP-deficient **RMA-S** tumor cells and TAP-deficient Con A blasts, whereas the corresponding TAP-expressing target cells were considerably less susceptible or resistant to lysis. The CTL epitopes recognized were expressed also by the human TAP-deficient cell line T2, transfected with appropriate **MHC** class I molecules. B6 mice immunized with B7-1-transfected TAP-deficient **RMA-S** cells were protected from outgrowth of a subsequent **RMA-S** tumor challenge. These findings are discussed in relation to the biochemical nature of **MHC** class I dependent CTL epitopes associated with impaired TAP function, as well as implications for immunotherapy and autoimmunity.

L5 ANSWER 4 OF 4 MEDLINE

ACCESSION NUMBER: 95045915 MEDLINE

DOCUMENT NUMBER: 95045915

TITLE: In vitro priming of cytotoxic T lymphocytes against poorly immunogenic epitopes by engineered antigen-presenting cells.

AUTHOR: Bellone M; Iezzi G; Manfredi A A; Protti M P; Dellabona P; Casorati G; Rugarli C

CORPORATE SOURCE: Istituto Scientifico H. San Raffaele, Milan, Italy.

SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1994 Nov) 24 (11) 2691-8. Journal code: EN5. ISSN: 0014-2980.

PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals; Cancer Journals

ENTRY MONTH: 199502

AB Cytotoxic T lymphocytes (CTL) recognize antigenic peptides presented by major histocompatibility complex class I (**MHC**-I) molecules on the surface of target cells. Optimal induction of CD8+ CTL depends on the amount of relevant peptide/**MHC**-I complexes and the presence of co-stimulatory molecules on antigen-presenting cells (APC). The antigen-processing defective mutant cell line **RMA-S**, when cultured at low temperature, expresses high amounts of **MHC** -I molecules that do not contain endogenously derived peptides. These "empty" **MHC**-I molecules can be stabilized by addition of **MHC**-binding peptides. **RMA-S** cultured at low temperatures with selected peptides have been used for in vitro CTL induction with conflicting results. **RMA-S** cells do not express detectable amounts of B7 co-stimulatory molecule. This could explain their unpredictable efficiency as APC. We have evaluated whether **RMA-S** cells, stably transfected with cDNA encoding for the human B7.1 molecule could provide effective co-stimulation for CD8+ T lymphocytes. **RMA-S**/B7 cells, loaded with different synthetic peptides, demonstrated a high and sometimes unique efficiency for in vitro primary CTL induction, even when "sub-optimal" antigen peptides were used. Most importantly, **RMA-S**/B7 cells pulsed with naturally processed peptides extracted from the poorly

immunogenic B16 melanoma cells were able to prime CD8+ cells against B16 melanoma. We conclude that the use of **RMA-S**/B7 cells as APC represents an ideal strategy for in vitro CTL immunization without prior in vivo priming. This system may also help to address the issue of the different contributions of co-stimulation and relative occupancy of **MHC**-I by single peptide epitopes in CTL priming.

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COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	82.13	82.28

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-1.67	-1.67

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